STATE OF MICHIGAN
IN THE SUPREME COURT

GALE BOERTMANN,

Plaintiff-Appellee,

-vs-

THE CINCINNATI INSURANCE COMPANY, a foreign corporation,

Defendant-Appellant.

Docket No. 142936
Court of Appeals docket no. 293835
Macomb Circuit Court
Hon. Peter J. Maceroni
LC No. 2008-003332-NF

BRIEF AMICUS CURIAE OF MICHIGAN ASSOCIATION FOR JUSTICE

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TABLE OF CONTENTS

INDEX OF AUTHORITIES

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STATEMENT OF QUESTIONS PRESENTED

I.

IS POST-TRAUMATIC STRESS DISORDER A "BODILY INJURY"?

Plaintiff-Appellee Gale Boertmann would answer "YES."

Defendant-Appellant Cincinnati Insurance Company would answer "NO."

The trial court answered "YES."

The Court of Appeals answered "YES."

Amicus Curiae Michigan Association for Justice answers "YES."

II.

DOES POST-TRAUMATIC STRESS DISORDER "ARISE OUT OF" WITNESSING THE DEATH OF A CLOSE RELATIVE IN A MOTOR VEHICLE ACCIDENT?

Plaintiff-Appellee Gale Boertmann would answer "YES."

Defendant-Appellant Cincinnati Insurance Company would answer "NO."

The trial court answered "YES."

The Court of Appeals answered "YES."

Amicus Curiae Michigan Association for Justice answers "YES."

INTEREST OF AMICUS CURIAE

The Michigan Association for Justice (MAJ) is an organization of Michigan lawyers engaged primarily in litigation and trial work. MAJ recognizes an obligation to assist this Court on important issues of law that would substantially affect the orderly administration of justice in the trial courts of this state.

STATEMENT OF FACTS

Amicus curiae Michigan Association for Justice accepts the statement of facts appearing in plaintiff-appellees Gale Boertmann's ("plaintiff") brief.

ARGUMENT I

POST-TRAUMATIC STRESS DISORDER IS A "BODILY INJURY."

(a)

Post-traumatic stress disorder is a recognized diagnosis with an objective physical component.

The idea that experiencing a traumatic event may damage a person who is not physically injured seems too obvious to require supporting authority. In 1980, however, psychiatry formally acknowledged the phenomenon. In its third Diagnostic and Statistical Manual of Mental Disorders ("DSM-III")[1], the American Psychiatric Association introduced the diagnosis "post-traumatic stress disorder" ("PTSD"). According to one authority, "[t]he initiation of PTSD in DSM-III in 1980 represented 'a paradigm shift in the conceptualization of post-trauma illness'. . ."[2] In 1994, a somewhat-revised definition appeared in the fourth edition, DSM-IV[3].

The most recent version of the DSM ("DSM-IV-TR"[4]), published in 2000, specifies six diagnostic criteria for PTSD. Among the relevant point are:

> A. The person has been exposed to a traumatic event . . .
>
> 1. The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
> 2. The person's response involved intense fear, helplessness, or horror. . .
>
> B. The traumatic event is persistently reexperienced [as]

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[1]American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, 3rd ed (Washington, DC: 1980). According to the APA, its DSM is "the standard classification of mental disorders used by mental health professionals in the United States." www.psychiatry.org.
[2] Anderson, *Post-traumatic stress disorder recognized in victims of many traumas*, 14(2) J Controversial Medical Claims 1 (2007).
[3]American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, 4th ed (Washington, DC: 1994).
[4]American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, 4th ed-text revision (Washington, DC: 2000). "DSM-V," a new edition of the manual, is scheduled for release in 2013. www.psychiatry.org.

1. Recurrent and intrusive distressing recollections of the event . . .
2. Recurrent distressing dreams of the event . . .
3. Acting or feeling as if the traumatic event were recurring . . .
4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
5. *Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event*

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness . . .

D. *Persistent symptoms of increased arousal* (not present before the trauma), as indicated by two (or more) of the following:

1. *Difficulty falling or staying asleep*
2. *Irritability or outbursts of anger*
3. *Difficulty concentrating*
4. *Hypervigilance*
5. *Exaggerated startle response*

E. Duration of the disturbance . . . more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. [DSM-IV-TR 309.81. Emphasis supplied.]

That is, even to be given a diagnosis of "post-traumatic stress disorder," a person must exhibit a physical reaction to reminders of the trauma ("physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event") and show two or more of a group of symptoms that include sleep disturbance; anger; concentration problems; being overly alert to potential threats ("hypervigilance"); and an increased level of reaction to unexpected stimuli ("exaggerated startle response").

The estimated incidence of PTSD varies. One review reported that "the estimated lifetime prevalence of PTSD among U.S. adults. . . is documented at 8% (10% in women, 5% in men) . . ."[5] According to a recent report, approximately 8 to 18% of trauma-exposed individuals

[5]Neria, DiGrande & Adams, *Posttraumatic stress disorder following the September 11, 2001, terrorist attacks*, 66(6) American Psychologist 429, 429 (2011). Another study puts the "lifetime prevalence as high as 6.8%." Woon & Hedges, *Gender does not moderate hippocampal volume*

develop PTSD.[6] It is much more common in women than men.[7] City residents have been found to be more likely to develop PTSD symptoms than those in suburban or rural environments.[8]

PTSD "is associated with lowered physical health status and a greater utilization of health care services for physical health problems"[9] and "health issues such as chronic pain, hypertension, coronary artery disease, thyroid disorder and other medical symptoms."[10] Greater incidence of asthma, heart disease, rheumatoid arthritis, metabolic syndrome, dementia and mortality have been observed in chronic PTSD populations.[11] PTSD is also a risk factor for suicide.[12]

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deficits in adults with posttraumatic stress disorder: A meta-analysis, 21(3) Hippocampus 243 (2011) (citing a 2005 National Comorbidity Survey Replication Study).

[6]Morris, Compas & Garber, *Relations among posttraumatic stress disorder, comorbid major depression, and HPA function: A systematic review and meta-analysis*, 32(4) Clinical Psychology Rev 301, 302 (2012) (citations omitted). Other reports indicate that in high-risk groups, such as victims of violent crimes or combat veterans, PTSD rates range from three to 58 percent. Anderson, *supra*.

[7]Lifetime prevalence rates of PTSD is twice as high females as males. Morris et al., *supra* at 304 (citations omitted). Women are twice as likely as men to develop PTSD during their lifetimes. Woon & Hedges, *supra* at 243 (citations omitted).

[8]Parto, Evans & Zonderman, *Symptoms of posttraumatic stress disorder among urban residents*, 199(7) J Nervous & Mental Diseases 436 (2011). See also Hunt, Martens & Belcher, *Risky business: Trauma exposure and rate of posttraumatic stress disorder in African American children and adolescents*, 24(3) J Traumatic Stress 365, 366-368 (2011).

[9]O'Donnell, Creamer, Elliott & Atkin, *Health costs following motor vehicle accidents: The role of posttraumatic stress disorder*, 18(5) J Traumatic Stress 557 (2005) (citations omitted).

[10]Parto et al. (citations omitted).

[11]Baker, Nievergelt & O'Connor, *Biomarkers of PTSD: Neuropeptides and immune signaling*, 62(2) Neuropharmacology 663 (2012) (citations omitted).

[12]Baker et al., *supra* at 667 (citations omitted).

(b)

PTSD can result from motor vehicle accidents.

In the case of motor vehicle accidents, the prevalence of posttraumatic stress disorder has been reported to vary widely. One study uses the range of 8.5% to 39%.[13] Another cited estimates of 10 and 46%.[14] Another study put it at 5.0% of men and 10.4% of women.[15] Women are more likely to develop PTSD after a motor vehicle accident than men.[16]

Several direct studies have been conducted. A leading British study found PTSD in 11% of motor vehicle accident survivors one year after the accident.[17] A U.S. study found 39.2% of patients at a large emergency department had diagnosable PTSD.[18] Another study found incidence of 34.4% at one month post-accident, diminishing to 18.2% at six months.[19] In Turkey, researchers found PTSD in 29.8%, 23.1% and 17.9% of 95 motor vehicle accident

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[13]Iteke, Bakare, Agomoh, Uwakwe & Onwukwe, *Road traffic accidents and posttraumatic stress disorder in an orthopedic setting in south-eastern Nigeria: A controlled study*, 19 Scandinavian J Trauma Resuscitation & Emergency Medicine 39 (2011) (citations omitted).

[14]Yaşan, Güzel, Tamam & Ozkan, *Predictive factors for acute stress disorder and posttraumatic stress disorder after motor vehicle accidents*, 42 Psychopathology 236 (2009) (citations omitted). The wide variation is likely due to differences in diagnostic tools (a number of different scales and interview protocols are in use) and study parameters as well as the wide variety of patient populations examined.

[15]Irish, Ostrowski, Fallon, Spoonster, van Dulmen, Sledjeski & Delahanty, *Trauma history characteristics and subsequent PTSD symptoms in motor vehicle accident victims*, 21(4) J Traumatic Stress, 377, 377 (2008) 188 adult MVA victims (98 men and 90 women) with a prior history of exposure to at least one potentially traumatic event).

[16]Fullerton, Ursano, Epstein, Crowley, Vance, Kao, Dougall, & Baum, *Gender differences in posttraumatic stress disorder after motor vehicle accidents*, 158(9) Amer J Psychiatry 1486 (2001) (122 subjects, one month after the accident; 18.8% of the men and 51.7% of the women developed PTSD).

[17]Mayou, Bryant & Duthie, *Psychiatric consequences of road traffic accidents*, 307 British Med J 647 (1993).

[18]Blanchard, Hickling, Taylor, & Loos, *Psychiatric morbidity associated with motor vehicle accidents*, 183(8) J Nervous & Mental Disease 495 (1995).

[19]Ursano, Fullerton, Epstein, Crowley, Kao, Kelley, Craig, Dougall & Baum, *Acute and chronic posttraumatic stress disorder in motor vehicle accident victims*, 156(4) Amer J Psychiatry 589 (1999)

victims at one, three and six months after the accident.[20] A survey of motor vehicle accident victims in Hong Kong one week, one month, three months, and six months after the accident found 5-20% had PTSD symptoms.[21] A study of 150 victims of motor vehicle accidents in Nigeria (which has poor roads and many accidents) found injured road traffic accident victims had higher prevalence of PTSD compared to those exposed to other traumas and nontrauma-exposed controls.[22]

The severity of the accident and the victim's injuries do not clearly predict the development of PTSD.[23] The condition can become long-term[24] and the cost of PTSD treatment adds to the expense of treatment of motor vehicle accidents.[25]

There is, then, an established basis for the treatment of PTSD arising out of motor vehicle accidents.

(c)

Research has established physiological components of PTSD.

As one author commented, "PTSD symptoms have been documented for centuries."[26] There is an extensive body of medical, psychological, psychiatric and sociological literature on PTSD, a full review of which would require an entire textbook.

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[20]Yasan et al. at 238.
[21]Wu & Cheung, *Posttraumatic stress after a motor vehicle accident: A six-month follow-up study utilizing latent growth modeling*, 19(6) J Traumatic Stress 923, 933 (2006).
[22]Iteke et al., *supra*. Female victims experienced PTSD more than the males.
[23]See, e.g., Fujita & Nishida, *Association of objective measures of trauma exposure from motor vehicle accidents and posttraumatic stress symptoms*, 21(4) J Traumatic Stress 425 (2008) (93 motor vehicle accident survivors in Japan, approximately five months after the incident; 49 followed up 14 months later).
[24]Mayou, Ehlers & Bryant, *Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study*, 40(6) Behaviour Research & Therapy 665 (2002) (3 years post-accident, 11% the participants met DSM-IV criteria for PTSD; participants meeting diagnostic criteria for PTSD at 1 year had a 47% chance of still suffering from PTSD at 3 years; there was also a 5% incidence of delayed onset of the disorder).
[25]See O'Donnell et al., *supra* at 560.

One study found that 35% of PTSD symptoms "are associated with physiological reactivity . . ."[27] PTSD patients experience dysfunctions of the autonomic nervous system, which regulates most physiological processes below the level of consciousness. PTSD patients show symptoms such as "hyperarousal, difficulty with responsiveness to stressors [and] impaired relaxation response."[28] The disorder may be understood as an impairment in unlearning of fear responses.[29] It is "extensively involved" some forms of learning and memory."[30]

The following is a summary of relevant recent findings on physiological markers, indices and, to a lesser extent, causes of PTSD.

i. Startle response

The "startle response" or "startle reflex" is an involuntary, primarily muscular, reaction to a sudden loud noise or similar event. It "consists of a bilateral generalized flexion reflex [i.e., contraction of groups of muscles], primarily in the head and shoulders."[31] It "is mediated by simple brainstem neural circuitry," [32] that is, it is controlled by a simple neural pathway located in the most primitive portion of the brain.

A review of the extensive scientific literature on that startle response is outside the scope of this brief, but it is well-established that increased startle response has been documented in

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[26]Neria, *supra* at 429.
[27]Grasso & Simons, *Electrophysiological responses to threat in youth with and without posttraumatic stress disorder*, 90 Biological Psychology 88, 88 (2012).
[28]Tan, Dao, Farmer, Sutherland & Gevirtz, *Heart rate variability (HRV) and posttraumatic stress disorder (PTSD): A pilot study*, 36(1) Appl Psychophysiol Biofeedback 27 (2011).
[29]Robinson & Shergill, *Imaging in posttraumatic stress disorder*, 24 Current Opinion in Psychiatry 29, 31 (2011).
[30]Woon & Hedges at 243.
[31]Siegelaar, Olff, Bour, Veelo, Zwinderman, van Bruggen, de Vries, Raabem, Cupido, Koelman & Tijssen, *The auditory startle response in post-traumatic stress disorder*, 174 Experimental Brain Research 1 (2006).
[32]Orr, Metzger & Pitman, *Psychophysiology of post-traumatic stress disorder*, 25(2) Psychiatric Clinics of North America 271, 277 (2002), citing Davis, *The mammalian startle response* in RC Eaton (ed.) Neural mechanisms of Startle (New York: Plenum, 1984) 287–351; Siegelaar et al, *supra* at 2.

patients with PTSD. As one reviewer noted, "[r]eports of exaggerated startle response in trauma survivors has a long history dating back to early observations of combat veterans."[33] The evidence of heightened acoustic startle reactivity is "compelling."[34]

Increased startle response has been observed in "male Vietnam combat veterans . . . male combat veterans from other wars . . . male police officers . . . female survivors of sexual assault . . . female survivors of childhood sexual abuse . . . as well as [in] studies of mixed trauma samples . . ."[35] The more limited research on female survivors is consistent.[36]

For example, Siegelaar et al., measured muscle activity in response to loud sounds in several muscles of the head and neck. In samples of patients with PTSD lasting at least three months, they found a significant difference between the PTSD subjects and a control group in one facial muscle.[37] Wessa et al.[38] compared a group of motor vehicle accident survivors from Germany with PTSD, an equal number without PTSD and normal controls as they were shown slides with positive, neutral and accident-related content; the negative slides PTSD group had higher startle responses to the accident-related slides than the non-PTSD group.[39]

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[33]Griffin, *A prospective assessment of auditory startle alterations in rape and physical assault survivors*, 21(1) J Traumatic Stress 91, 91 (2008) (citations omitted).
[34]Id.
[35]Griffin, *supra* at 97.
[36]Griffin, *supra* at 98. See also, e.g., Butler, Braff, Rausch, Jenkins, Sprock & Geyer, *Physiological evidence of exaggerated startle response in a subgroup of Vietnam veterans with combat-related PTSD*, 147(10) Am J Psychiatry 1308 (1990) (veterans with PTSD showed increased startle reactivity to the loud noise in comparison with control subjects).
[37]174 Experimental Brain Research 5.
[38]Wessa, Karl & Flor, *Central and peripheral psychophysiological responses to trauma-related cues in subclinical posttraumatic stress disorder: A pilot study*, 167(1) Experimental Brain Research 56 (2005).
[39]Id. at 60.

See also, e.g., Elsesser et al.[40] ("some evidence of exaggerated startle reactions in PTSD patients but not in recent trauma victims"); Jovanovic et al. (veterans of the Serbo–Croatian war with PTSD for up to 10 years were less able to get used to an arousing stimulus; data "suggest alterations in parasympathetic activity in addition to the sympathetic system hyper-arousal")[41]; Griffin, *supra* (women victims of rape or other assault showed with PTSD showed greater eyeblink response at six months than those without PTSD); Shalev et al.[42] (results supported the conclusion that abnormally elevated reactions "develops along with PTSD"); Hauschildt et al.[43] (PTSD subjects' heart rate differed from other trauma-exposed and normal control subjects when viewing video scenes of various emotional contents, suggestive of parasympathetic impact); Tan et al., *supra* (heart rate variability, reflecting ability to adaptively cope with stress, found significant difference between veterans with combat-related PTSD and other veterans)[44].

Some authors have specifically examined PTSD in motor vehicle accident victims. Rabe et al. did EKG studies of 73 survivors of motor vehicle accidents at least six months later and compared them to 27 healthy controls.[45] The experimenters measured the subjects' heart rates in response to positive, negative and neutral pictures. There was an increase in heart rate during

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[40]Elsesser, Sartory & Tackenberg, *Attention, heart rate, and startle response during exposure to trauma-relevant pictures: A comparison of recent trauma victims and patients with posttraumatic stress disorder*, 113(2) J Abnorm Psychol 289, ___ (2001).
[41]Jovanovic, Norrholm, Jambrošić Sakoman, Esterajher & Kozarić-Kovačić, *Altered resting psychophysiology and startle response in Croatian combat veterans with PTSD*, 71 Internat'l J Psychophysiology 264, 268 (2009).
[42]Shalev, Peri, Brandes, Freedman, Orr & Pitman, *Auditory startle response in trauma survivors with posttraumatic stress disorder: A prospective study*, 157(2) Amer J Psychiatry 255, 259 (2000).
[43]Hauschildt, Peters, Moritz & Jelinek, *Heart rate variability in response to affective scenes in posttraumatic stress disorder*, 88 Biological Psychology 215, 219-220 (2011).
[44] 36(1) Appl Psychophysiol Biofeedback 27.
[45]Rabe, Dörfel, Zöllner, Maercker & Karl, *Cardiovascular correlates of motor vehicle accident related posttraumatic stress disorder and its successful treatment*, 31(4) Applied Psychophysiological Biofeedback 315 (2006).

the trauma-related condition for PTSD participants in comparison to the two control groups. The PTSD patients had a higher baseline heart rate. It was significantly associated with PTSD severity.[46]

ii. Changes in brain structures

The human brain is an enormously complex organ, composed of a multitude of specialized but interrelated parts. See Stedman's, *supra* at 251 for an overview. Abnormalities in the limbic system, a group of structures deep in the brain associated with processing external stimuli and emotional (feeling) responses, have been implicated in PTSD.

Hippocampus. The hippocampus is a structure where "a massive convergence of cortical neurons from various association areas of the brain" takes place.[47] It resembles "an old-fashioned central telephone switchboard."[48] "It has . . . been suggested that the hippocampus is the structure particularly associated with memories of conscious experience."[49] It is involved in learning and memory.

In a lengthy review of the relevant literature to 2000, McEwen[50] pointed to numerous studies showing that the hippocampus is sensitive to adrenal steroid hormones, produced in response to stress, suggesting a connection between stress and changes in this area of the brain.[51] More recently, Karl et al.[52] after reviewing a large number of studies, found "reliable evidence"

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[46] *Id.* at 320.
[47] Grossman, Buchsbaum & Yehuda, *Neuroimaging studies in post-traumatic stress disorder*, 25(2) Psychiatric Clinics of North America 317, 318 (2002).
[48] *Id.*
[49] Brewin, *A cognitive neuroscience account of posttraumatic stress disorder and its treatment*, 39 Behaviour Research and Therapy 373, 378 (2001) (citation omitted).
[50] McEwen, *The neurobiology and neuroendocrinology of stress: Implications for post-traumatic stress disorder from a basic science perspective*, 25(2) Psychiatric Clinics N America 469, 476-481 (2002).
[51] *Id.* at 483.
[52] Karl, Schaefer, Malta, Dorfel, Rohleder & Werner, *A meta-analysis of structural brain abnormalities in PTSD*, 30 Neuroscience and Biobehavioral Reviews 1004 (2006).

of reduced size of the hippocampus in PTSD patients. A meta-analysis (review of many other studies combined) also showed differences in cortisol levels between individuals with PTSD and those who had not been exposed to trauma.[53] Another review of 23 published studies, found bilateral [both halves of the brain] hippocampal volume reduction in PTSD subjects compared with control subjects.[54]

Amygdala. Another brain structure, the amygdala, has also shown changes in PTSD patients. The amygdala "is thought to assess and assign emotional valence to somatic, visceral and olfactory sensory input."[55] That is, it is involved in associating emotions with the perception of stimuli. It "is responsible for initiating a variety of hard-wired responses to threat including release of stress hormones, activation of the sympathetic nervous system, and behavioural responses such as fight/flight and freezing."[56] A review of studies of hippocampal and amygdaloid activity in the PTSD patients found several that demonstrated changes in activity in both these regions.[57]

Cortical regions. The limbic system interacts with other areas of the brain. PTSD and multiple regions of the cortex (where thinking, reasoning and other higher-order functions originate) has been studied.

A recent review of multiple functional MRI studies including thousands of measurements confirmed that "the amygdala, the anterior cingulate, and the middle frontal gyrus [are] regions

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[53]Morris et al., *supra* at 302.
[54]Woon & Hedges, *supra* at 246-247. They did not, however, find a significant sex difference.
[55]Stedman's, p 68.
[56]Brewin, *supra* at 376-377 (citation omitted).
[57]Grossman et al, *supra* at 329.

that were differentially activated between PTSD and non-PTSD individuals."[58] Another study found a thinner prefrontal cortex in war veterans with chronic PTSD.[59]

Kroes et al.[60] did MRI studies of patients with PTSD, depression and control subjects. They observed similar reductions in brain volume in the prefrontal cortex (part of the brain associated with higher cognitive functioning) in both the patient groups, although they did not find a distinct pattern for PTSD alone.[61] Schuff et al.[62] found evidence of abnormalities in the frontal cortex, limbic system and specific hippocampal subfields." Kroes et al.[63] found that reduced volume in the grey matter (nerve cells) in a number of areas was associated with overall increases in reexperiencing "flashback" scores.[64]

An unusual study reexamined veterans (25 with PTSD and 22 controls) who had participated in an imaging study two years earlier.[65] The veterans who had no improvement in symptoms after two years showed significantly greater atrophy (deterioration) of tissue in

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[58]Simmons & Matthews, *Neural circuitry of PTSD with or without mild traumatic brain injury: A meta-analysis*, 62(2) Neuropharmacology 598, 600 (2012).
[59]Eckart, Stoppel, Kaufmann, Tempelmann, Hinrichs, Elbert, Heinze & Kolassa, *Structural alterations in lateral prefrontal, parietal and posterior midline regions of men with chronic posttraumatic stress disorder*, 36(3) J Psychiatry & Neuroscience (2011) (Kurdish war veterans).
[60]Kroes, Rugg, Whalley & Brewin, *Structural brain abnormalities common to posttraumatic stress disorder and depression*, 36(4) J Psychiatry & Neuroscience 256 (2011).
[61]It is not surprising that many patients with PTSD also have symptoms of depression, but the two disorders are distinct. As one group noted, "[a] problem in the study of PTSD is its high comorbidity with depression and alcohol abuse." Canive et al., *infra*, at 514.
[62]Schuff, Zhang, Zhan, Lenoci, Ching, Boreta, Mueller, Wang, Marmar, Weiner & Neylan, *Patterns of altered cortical perfusion and diminished subcortical integrity in posttraumatic stress disorder: A MRI study*, 54(Jan) Neuroimage S62 (2011).
[63]Kroes, Whalley, Rugg & Brewin, *Association between flashbacks and structural brain abnormalities in posttraumatic stress disorder*, 26(8) European Psychiatry 525 (2011)
[64]Kroes et al. 2 at 528.
[65]Cardenas, Samuelson, Lenoci, Studholme, Neylan, Marmar, Schuff & Weiner, *Changes in brain anatomy during the course of posttraumatic stress disorder*, 193(2) Psychiatry Research: Neuroimaging 93 (2011).

multiple areas of the brain[66] in comparison to subjects who did not have PTSD.[67] The results suggest "ongoing brain injury" and potentially greater risk for cognitive impairment and dementia in later life.[68]

Other studies reported changes in nearby areas of the brain that have been implicated in extinction (decrease of) the fear response to stimuli.[69]

iii. Neuroendocrine changes.

The "stress response system" is an important part of allowing organisms to accommodate to changing conditions in their environment.[70] In 1997, one authority noted that "[s]ince World War I, numerous clinicians and researchers have described signs and symptoms of sympathetic nervous system dysregulation in combat veterans who suffer from what is currently termed posttraumatic stress disorder."[71] Research dates to 1918.[72]

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[66]Insula, anterior cingulate, dorsolateral prefrontal cortex, anterior temporal lobe, and extrastriate cortex.

[67]Cardenas et al. at 96-97.

[68]Cardenas et al. at 99.

[69]Grossman et al, *supra* at 329-330, 332. As one group stated, "a convergence of findings from functional neuroimaging investigations in clinical populations supports a neurocircuitry model of PTSD characterized by abnormally elevated amygdala activity coupled with deficient regulation by prefrontal cortical structures." Murrough, Huang, Hu, Henry, Williams, Gallezot, Bailey, Krystal, Carson & Neumeister, *Reduced amygdala serotonin transporter binding in posttraumatic stress disorder*, 70(11) Biological Psychiatry 1033, 1033 (2011) (reporting evidence of "altered amygdala functioning that in turn drives increased anxiety and vulnerability to the effects of stress and trauma;" id. at 1036). See also Mezey & Robbins, *Usefulness and validity of post-traumatic stress disorder as a psychiatric category*, 323.7312 British Medical Journal 561 (2001). See also, e.g., Canive, Lewine, Orrison, Edgar, Provencal, Davis, Paulson, Graeber, Roberts, Escalona & Calais, *MRI reveals gross structural abnormalities in PTSD*, 821 Annals of NY Academy of Sciences 512, 514-515 (1997) (more indications of damage to white matter of brain in PTSD patients than in normal population).

[70]Morris et al at 302.

[71] Southwick, Morgan, Bremner, Grllon, Krystal, Nagy & Charney, *Noradrenergic alterations in posttraumatic stress disorder*, 821 Annals of the NY Acad of Sciences 125, 125 (1997).

[72] Southwick et al.

The hypothalamic–pituitary–adrenocortical ("HPA") axis is one of three major systems activated as part of the stress response.[73] It plays an important role in both the stress response and the maintenance of homeostasis.[74]

Cortisol. Cortisol is "a steroid hormone or glucocorticoid produced by the adrenal gland via control from the hypothalamus."[75] It "is regarded as a key component of the physiological response to fear and stress in studies on PTSD."[76] It "is one of the primary modulators of the stress response" and is also involved in the consolidation of memory (the formation of permanent memories of sensory input).[77]

"Although elevated cortisol levels are adaptive in the short-run, prolonged activation of the HPA-axis can have adverse effects."[78] For example, "[e]ven transient elevations in plasma cortisol have been found to damage the hippocampus in nonhuman species, resulting in loss of neurons."[79]

"[T]he majority of the evidence" supports the conclusion that cortisol levels in PTSD "are directionally different from those observed in acute and chronic stress and major depression" and that the HPA axis[80] "appears to be regulated differently."[81]

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[73] Morris et al., *supra* at 302 (citation omitted).
[74] For a summary of the physiology of the HPA feed-forward function, see Morris et al at 302.
[75] Luo, Hu, Liu, Ma, Guo, Qiu, Wang, Wang, Zhang, Zhang, Hannum, Zhang, Liu & Li, *Hair cortisol level as a biomarker for altered hypothalamic-pituitary-adrenal activity in female adolescents with posttraumatic stress disorder after the 2008 Wenchuan earthquake*, 72(1) Biological Psychiatry 65, 65 (2012).
[76] Luo et al., *supra* at 65.
[77] McFarlane, Atchison & Yehuda, *The acute stress response following motor vehicle accidents and its relation to PTSD*, 821 Annals of the NY Academy of Sciences 437, 437 (1997).
[78] Morris et al. at 303 (citation omitted).
[79] Murburg, *supra*, 821 Annals of NY Acad of Science, 355.
[80]The hypothalamic-pituitary-adrenal ("HPA") axis refers to the interactive nature of the hypothalamus, pituitary and thyroid glands in the regulation of thyroid hormone. Stedman's, *supra*, at 937. The thyroid is implicated in metabolism.

Adrenaline and noradrenalin. "In an acutely stressful situation, the CNS release of norepinephrine allows for the direction of attention to relevant stimuli and participates in the activation of pathways for sympathoadrenal responses."[82] The result "alter[s] heart rate and blood pressure and mobilize energy substrates also made more available by cortisol."[83] Southwick et al., *supra*, concluded that "strong evidence for noradrenergic dysregulation in a subgroup of individuals with chronic PTSD."[84] Increased adrenaline could explain several symptoms of PTSD, including hypervigilance, exaggerated startle, and insomnia.[85] Norepinephrine "also has been implicated in the reexperiencing symptoms" of PTSD.[86]

Other hormones. There is an "emerging awareness" that "immune system cells and molecules are physiological participants in the response to psychological stress, unassociated with infectious or physical injury."[87] "PTSD is among the disorders that show evidence of enhanced CNS reactivity, increased inflammation and deleterious health consequences."[88] More severe disturbances in neuroendocrine and cytokine mediators "predict more severe PTSD

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[81] Yehuda, *Current status of cortisol findings in post-traumatic stress disorder*, 25(2) Psychiatric Clinics of North America 341, 342 (2002). See also Grossman et al, *supra* at 318-319; McFarlane, *supra* at 440 (mean cortisol levels of subjects who had PTSD 6 months after the accident were significantly lower than those with depression); Kellner, Baker & Yehuda, *Salivary cortisol and PTSD symptoms in Persian Gulf war combatants*, 821 Annals of the NY Acad of Sciences 442, 443 (1997) (pilot study finding lower cortisol levels in soldiers with PTSD symptoms).

[82] Murburg, *The psychobiology of posttraumatic stress disorder: An overview*, 821 Annals of the NY Acad of Sciences 352, 355 (1997). For a more detailed description, see Baker et al., infra at 664.

[83] Murburg.

[84] Southwick et al., *supra* at 134.

[85] Southwick et al., *supra* at 134.

[86] Southwick et al., *supra* at 134.

[87] Baker et al., *supra* at 663.

[88] Baker et al., *supra* at 664.

symptoms .. . ."[89] "[A]lterations have been reported in . . . catecholamine, opiate, and thyroid systems as well as in the HPA axis."[90]

iv. EEG measurements

The central nervous system (CNS) "recognizes an environmental stressor as a potential threat" within about .2 seconds [200 milliseconds], and triggers a response from the autonomic nervous system.[91] "Event-related potentials" are changes in the brain in response to external stimuli that can be measured with EEG equipment. They are "a fruitful method of examining information processing."[92]

Most studies use repeated auditory or visual stimuli; record the responses electronically; and use a computer program to average the response amplitude (how much of a response was produced) over a specific amount of time after the stimulus is presented. A very recent review articles noted that "[c]onverging neuroimaging research suggests altered emotion neurocircuitry in individuals with posttraumatic stress disorder."[93]

Javanbakht et al.[94] reviewed 36 studies attempting to document PTSD via event-related potentials. A significant number of studies found increased responses at the 300 millisecond [.3 seconds] point for stimuli that were trauma-related or otherwise unpleasant. "[S]ubjects with PTSD show sensitization and impaired habituation to the stimuli which represent the traumatic stimuli."

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[89] Baker et al., *supra* at 668.
[90] Southwick et al.
[91] Grasso & Simons, *supra* at 88.
[92] Grasson & Simons, *supra* at 88.
[93]Garfinkel, King, Liberzon, Sripada, Sripada & Wang, *Altered resting-state amygdala functional connectivity in men with posttraumatic stress disorder*, 37(4) Journal of Psychiatry and Neuroscience 241 (2012).
[94]Javanbakht, Liberzon, Amirsadri, Gjini & Boutros, *Event-related potential studies of post-traumatic stress disorder: A critical review and synthesis*, Biology of Mood & Anxiety Disorders (2011).

In a study of soldiers returned from Afghanistan who had PTSD, the subjects' brain activity was measured using functional magnetic resonance imaging (fMRI) while they did a short-term memory task.[95] The experimenters found increased activity in several parts of the brain that process emotion. The results were consistent with the clinical picture of PTSD.[96]

A recent study of a different evoked potential compared 22 people with diagnosable PTSD, resulting from many causes, including motor vehicle accidents, to 35 without it.[97] The subjects with PTSD "[found] threatening outcomes more probable."[98] The data provide "direct neural evidence of bias in those with PTSD."[99] Another study, of women with PTSD as a result of childhood trauma, also reported differences in the medial prefrontal cortex (part of the brain involved in higher-level processing and appreciation of social stimuli) in response to anxiety-provoking events.[100]

In the case of motor vehicle accident victims, brain electrical activity during rest and during the presentation of emotional (neutral, positive, negative, and trauma-related) pictures in patients with PTSD found the response of the PTSD patients' right hemisphere was significantly different from that of other groups of subjects.[101]

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[95]Bates, *Imaging ties PTSD to altered brain function*, 42(12)Internal Medicine News 40 (2009).
[96]*Id.*
[97]Kimble, Batterink, Marks, Ross & Fleming, *Negative expectancies in posttraumatic stress disorder: Neurophysiological (N400) and behavioral evidence*, 46(7) J Psychiatric Research 849 (2012).
[98] Kimble et al., *supra* at 853.
[99]Kimble et al., *supra* at 853.
[100]Frewen, Dozois, Neufeld, Lane, Densmore, Stevens, & Lanius, Emotional numbing in posttraumatic stress disorder: *A functional magnetic resonance imaging study*, 73(4) J Clin Psychiatry 431 (2012).
[101]Rabe, Beauducel, Zöllner, Maercker & Karl, *Regional brain electrical activity in posttraumatic stress disorder after motor vehicle accident*, 115(4) J Abnormal Psych 687 (2006).

v. MRI studies

Functional MRI studies have identified differences in the amygdala; the insula[102]; and the hippocampus.[103] These authors found differences in the amygdala even when the subjects were not engaged in a perceptual task, concluding "[t]hese findings suggest abnormalities in emotion generation and regulation circuits."[104] See also, e.g., Aupperle et al.[105] (neuroimaging showed significant difference in anticipation of negative emotional images in women with PTSD).

In short, studies of how the brain works and what it looks like, as well as assessments of hormonal changes related to brain activity, clearly show a physiological substrate for the feelings and behaviors that make up "post-traumatic stress disorder."

(c)

A no-fault insurer is liable for treatment of PTSD

MCL 500.3105(1) provides that "an insurer is liable to pay benefits for accidental bodily injury arising out of the ownership, operation, maintenance or use of a motor vehicle as a motor vehicle." MCL 500.3107(1)(a) provides that "personal protection insurance benefits are payable for . . . all reasonable charges incurred for reasonably necessary products, services and accommodations for an injured person's care, recovery, or rehabilitation."

As described *supra*, there is extensive evidence that PTSD is a behavioral and psychological manifestation of multiple underlying physiological changes to the structure and

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[102]An area "responsible for interoception, disgust, emotion processing, emotion recall and anticipation of aversive stimuli." Garfinkel et al., *supra*.

[103] *Id.*

[104]*Id.* The same study reported differences in the relationship between the amygdala and the prefrontal cortex between groups, suggesting "a deficit in automatic emotion regulation or a lack of cognitive control over emotion."

[105]Aupperle, Allard, Grimes, Simmons, Flagan, Behrooznia, Cissell, Twamley, Thorp, Norman, Paulus & Stein, *Dorsolateral prefrontal cortex activation during emotional anticipation and neuropsychological performance in posttraumatic stress disorder*, 69(4) Archives of Gen Psychology, 360, 366 (2012).

chemistry of the brain. It is, then, a "bodily injury" within the meaning of §3105(1). Payment for "reasonably necessary products, services and accommodations" for treatment of PTSD arising out of a motor vehicle accident, therefore, is required under the no-fault act.

ARGUMENT II

POST-TRAUMATIC STRESS DISORDER "ARISES OUT OF" WITNESSING THE DEATH OF A CLOSE RELATIVE IN A MOTOR VEHICLE ACCIDENT.

(a)

Witnessing a traumatic event is a recognized criterion for the diagnosis of post-traumatic stress disorder.

According to DSM-IV, *supra*, the first criterion for the diagnosis of PTSD is that the person "has been exposed to a traumatic event in which [he or she] . . . experienced, *witnessed, or was confronted* with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others" (emphasis supplied).

That is, a diagnosis of PTSD does not require that the patient him or herself have been the victim of "an event . . . that involved . . . death or serious injury . . ." in order for a professional to reach a diagnosis of PTSD. Thus, the viewing of the death of a relative in a motor vehicle accident is sufficient to satisfy criterion A(1) of the diagnosis under DSM-IV.

(b)

Research supports the existence of PTSD among those who witness traumatic events.

"[W]itnessing violence and trauma to others has been shown to be associated with PTSD in descriptive and epidemiologic studies."[106] Under DSM-III, "mere knowledge of the exposure

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[106]Carson, Paulus, Lasko, Metzger, Wolfe, Orr & Roger K. Pitman, *Psychophysiologic assessment of posttraumatic stress disorder in Vietnam nurse veterans who witnessed injury or death*, 68(5) J Consulting & Clinical Psychology 890, 890 (2000).

of a loved one to a traumatic event can be traumatizing as well."[107] A variety of studies support the proposition that PTSD can result from witnessing a trauma or otherwise indirectly experiencing it.

i. Physical and physiological evidence

In a leading paper, Carson et al.[108], did an intensive study of nurses who had served during the Vietnam war. Many of the nurses formed "strong emotional bonds" with the patients. About half had PTSD. The experimenters measured heart rate, skin conductance, and electromyograms of two facial muscles while the subjects listened to "scripts" related to witnessing a traumatic Vietnam experience, other stressful events, a positive event and a neutral one. In a control sequence, they listened to "scripts" that were not Vietnam-related or were Vietnam-related but not traumatic. The subjects with PTSD "had overall larger responses than non-PTSD participants, and participants' responses were larger during personal versus standard Vietnam imagery."[109] The authors concluded, "[t]he present results provide psychophysiologic support for the proposition that *witnessing death or serious injuries is a highly stressful experience that is associated with the development of PTSD* in some individuals."[110]

Luo et al.[111], studied hair cortisol seven months after an earthquake in 12-15 year old female survivors with and without PTSD and nontraumatized controls. Almost one-third of the PTSD subjects had witnessed someone die; more than 75% had witnessed another child's being buried or severely injured.[112] Using a recognized technique for measuring cortisol in scalp hair,

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[107]Figley & Kleber, *Beyond the "victim": secondary traumatic stress*, in R J Kleber, C R Figley & B P R Gersons (eds), *Beyond Trauma*, Plenum Press, NY, USA (1995) 77.
[108] 68(5) J Consulting & Clinical Psychology, *supra*.
[109] *Id.* at 893.
[110]*Id.* at 894 (emphasis supplied).
[111] 72(1) Biological Psychiatry, *supra*.
[112]72(1) Biological Psychiatry 67.

Luo et al found significant differences between the PTSD subjects and the non-PTSD and control girls.[113]

ii. Witnessing death in an ICU.

A number of researchers have examined the effects on relatives, or others, who witnessed the death of a hospital patient. Symptoms of posttraumatic stress disorder "are common among family members of patients who die in the ICU."[114]

A group of Indian researchers studied the incidence of PTSD in relatives of trauma patients admitted to the ICU of a hospital in India.[115] Using a paper-and-pencil assessment, the authors found 79% showed "severe psychological distress" seven days after admission. Of those who could be followed up two years later, many showed signs of PTSD.[116]

In France, about one-third of the relatives of ICU patients who had died reported symptoms diagnosable as PTSD 90 days later.[117] "[C]linically meaningful post-traumatic stress reaction was common and was the strongest risk factor was sharing in end-of-life decisions for the relative in the ICU."[118]

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[113] *Id.* at 67-68.
[114] Kross, Engelberg, Gries, Nielsen, Zatzick & Curtis, *ICU care associated with symptoms of depression and posttraumatic stress disorder among family members of patients who die in the ICU*, 139(4) Chest 795 (2011).
[115] Aigolikar, Ambike, Husainy, Kulkarni, Pillai & Vaidya, *The prevalence of post-traumatic stress disorder symptoms in relatives of severe trauma patients admitted to the intensive care unit*, 10(3) Indian Journal of Critical Care Medicine (2006).
[116] *Id.*
[117] Azoulay, Pochard, Kentish-Barnes, Chevret, Aboab, Adrie, *Risk of post-traumatic stress symptoms in family members of intensive care unit patients*, 171(2) Am J Respir Crit Care Med 987 (2005).
[118] *Id.*

Kross et al.[119], interviewed 226 family members of patients who had died in the ICU at least six months earlier. Family members who were present at the time of death reported higher symptoms of PTSD.[120]

See also Jones et al.[121] (a high percentage of relatives experience acute post-traumatic stress reactions during the first few weeks after ICU is a risk factor for the development of later PTSD).

iii. Witnessing assisted suicide.

Assisted suicide "can be considered an unnatural death," which is a risk factor for PTSD in family members.[122] Wagner et al.[123] studied a total of 85 people who had been present during the assisted suicide of a relative or friend in Switzerland, which permits assisted suicide, about 1½ years after the death. Of the respondents, 13% had PTSD and another 6.5% showed symptoms but did not meet the threshold for diagnosis.[124] They concluded, "[w]itnessing the unnatural death of a significant person thus seems to have a strong impact on the bereaved, which may lead to severe mental health problems at 14 to 24 months post-loss."[125]

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[119] 139(4) Chest 795, *supra.*
[120] 139(4) Chest 799. Families of older patients reported fewer symptoms. *Id.* at 799.
[121] Jones, Skirrow, Griffiths, Humphris, Ingleby & Eddleston, *et al.*, *Post-traumatic stress disorder-related symptoms in relatives of patients following intensive care.* 30 Intensive Care Med 456, 459 (2004).
[122] Wagner, Müller & Maercker, *Death by request in Switzerland: Posttraumatic stress disorder and complicated grief after witnessing assisted suicide*, ___ European Psychiatry ___ (2011).
[123] *Id.*
[124] *Id.* at *3. The age of the deceased person was a significant variable; where the deceased was younger, the respondent was more likely to have developed PTSD. *Id.* at *3, *5.
[125] *Id.* at *5.

iv. Witnessing other deaths and injuries.

A Michigan study investigated the effect of witnessing CPR on a relative in uncontrolled (out-of-hospital) settings.[126] Adult relatives (primarily spouses and adult children) of Beaumont Hospital patients were interviewed from 10 weeks to 5 months after the relative's death and compared with a control group who had not witnessed an attempt at resuscitation. Those who had witnessed the attempt had PTSD symptom scores almost twice as high as the control group; almost one-third of this group had enough symptomatology to be termed "clinically significant."[127]

In Iran, where public hangings are permitted, Attari et al. studied 200 primary school children (aged 7-11) who witnessed a hanging near their school.[128] Three months later, 52% showed at least one of the symptoms of PTSD and 12% fit the criteria for chronic PTSD.[129]

In a study conducted in 1995, researchers in Alabama collected data from 71 mostly-female employees of abortion clinics to assess the effect of their exposure to violence.[130] Most of the subjects witnessed episodes of violence rather than being participants themselves. More than 60% of the subjects had enough symptoms of PTSD to meet the diagnostic criteria.[131]

In a study of Australian children aged 6-12 who had been witnesses to violence (some of it life-threatening) against their mothers by a father or stepfather, all the children who had witnessed violence showed some symptoms of PTSD; almost half had a severe level of

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[126]Compton, Grace, Madgy & Swor, *Post-traumatic stress disorder symptomatology associated with witnessing unsuccessful out-of-hospital cardiopulmonary resuscitation*, 16(3) Academic Emergency Medicine 226 (2009).

[127]*Id.* at 228-229.

[128]Attari, Dashty & Mahmoodi, *Post-traumatic stress disorder in children witnessing a public hanging in the Islamic Republic of Iran*, 12(1-2) East Mediterr Health J 72 (2006).

[129]*Id.*

[130]Fitzpatrick & Wilson, *Exposure to violence and posttraumatic stress symptomatology among abortion clinic workers*, 12(2) J Traumatic Stress 227 (1999).

[131]*Id.* at 236, 238, 239. Respondents witnessing more violence reported more PTSD symptoms. *Id.* at 238.

symptomatology.[132] The authors concluded that "witnessing domestic violence is a stressor comparable in impact to the direct experience of a violent or abusive act."[133]

PTSD symptoms are relatively common in parents of children with serious medical conditions, including traumatic injury.[134] In a study of 99 parents of 12-18 year-olds, of which 42% of the injuries were from motor vehicle or motorcycle accidents, up to a year after a traumatic injury, a "significant subgroup" showed symptoms of PTSD as much as a year later.[135]

In children, "research has "demonstrated a distinct and detectable PTSD constellation . . . after traumatic experiences that involve a real threat to the physical integrity of the self or significant others."[136] A large cohort (232 children 1.5 to 5 years of age) of Israeli children living at the border of the Gaza Strip and exposed to daily rockets and terrorist attacks were observed with their mothers and compared with nonexposed matched controls. Almost 40% met the criteria for PTSD.[137]

v. Witnessing motor vehicle accidents.

Motor vehicle accidents are the main cause of PTSD in industrialized countries.[138] As one study stated, "[t]here is a wealth of evidence which suggests that significant posttraumatic

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[132]Kilpatrick & Williams, *Post-traumatic stress disorder in child witnesses to domestic violence*, 67(4) Am J Orthopsychiatry 639 (1997).

[133]*Id.*

[134]Martin-Herz, Rivara, Wang, Russo & Zatzick, *Predictors of parental posttraumatic stress disorder symptoms in the year after adolescent traumatic injury*, 12(3) Academic Pediatrics 198 (2012).

[135]*Id.* at 202. Unfortunately, the study did not distinguish among parents who witnessed the injury and those who did not.

[136]Feldman & Vengrober, *Posttraumatic stress disorder in infants and young children exposed to war-related trauma*, 50(7) J American Academy Of Child & Adolescent Psychiatry 645, 646 (2011) (citations omitted).

[137] *Id.* at 649, 654.

[138]Allenou, Olliac, Bourdet-Loubère, Brunet, David, Claudet, Lecoules, Roullet, Bui & Birmes, *Symptoms of traumatic stress in mothers of children victims of a motor vehicle accident*, 27(7) Depression and Anxiety 652, 652 (2010) (citations omitted).

distress is not limited to the victim alone."[139] Younger witnesses and females are more likely to experience symptoms of PTSD than older witnesses and males.[140]

Tierens et al.[141] studied Flemish high school students who had witnessed an auto accident, most of which had caused an injury. There were statistically significant differences between witnesses and the control group in their report of behavioral problems.[142] Witnesses, however, reported fewer trauma symptoms compared to injured victims.[143]

A French study found that 20% of mothers whose child had suffered a motor vehicle accident "presented significant PTSD symptoms,"[144] although the report did not separate those who saw the accident from those who did not. Parents whose children (aged 8-17) had been involved in a motor vehicle accident (include motor vehicle/pedestrian and motor vehicle/ bicycle accidents) one week later, with a follow-up at five weeks post-accident, were assessed. About one-quarter of the parents had witnessed the child's injury. Almost 22% of the parents (18% of the mothers and 4% of the fathers) who were available for the follow-up showed evidence of PTSD at the five-week assessment.[145]

vi. Mass trauma

A review of studies of PTSD in New Yorkers after September 11[146] reported that "[t]he most consistently documented correlates of PTSD across studies of 9/11-related PTSD were

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[139]Lerias & Byrne, *Vicarious traumatization: Symptoms and predictors*, 19(3) Stress & Health 129, 129 (2003).
[140]*Id.* at 134.
[141]Tierens, Bal, Crombez, Loeys, Antrop & Deboutte, *Differences in posttraumatic stress reactions between witnesses and direct victims of motor vehicle accidents*, 25 J Traumatic Stress 280 (2012).
[142]*Id.* at 283.
[143]*Id.* at 284.
[144]Allenou, *supra* 652, citing Unité de Recherche Interdisciplinaire OCTOGONE, CERPP, Université Toulouse le Mirail, Toulouse, France.
[145]Allenou et al., *supra* at 654-655.
[146]Neria et al., 66(6) American Psychologist, *supra*,

based on exposure to the event."[147] In particular, "loss of life of significant others, physical injury, and immediate risk of life were especially predictive of PTSD."[148]

A telephone survey of about 1000 Manhattan residents who lived near the World Trade Center during the fall of 2001 found a significant incidence of PTSD symptoms; PTSD was higher among the persons who were most directly exposed to the attacks or their consequences.[149]

A web-based survey of New Yorkers about 2 weeks after the September 11 attacks and again at two and six months compared a large (2000+) sample to over 900 controls.[150] The stress symptoms of individuals indirectly exposed resembled those that would that would be expected of symptoms reported by direct survivors.[151]

vii. Secondary trauma.

Several groups have found PTSD in soldiers or relief workers in areas of mass casualties. Dirkzwager et al.[152] found increased incidence of PTSD in family members of Dutch peace-keeping soldiers who had returned from various peacekeeping missions since 1975.

Litz et al.[153] studied a large group of soldiers who were assigned to "peacekeeping" in Somalia, which included both quasi-military and "humanitarian" functions. A "small but significant percentage of participants appeared to have PTSD following their service there."[154]

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[147] *Id.* at 440.
[148] 66(6) American Psychologist 440 ("strong evidence for a probable association between indirect exposure and PTSD).
[149] Galea, Ahern, Resnick, Kilpatrick, Bucuvalas, Gold & Vlahov, *Psychological sequelae of the September 11 terrorist attacks in New York City*, 346(13) N Engl J Med 982 (2002).
[150] Suvak, Maguen, Litz, Cohen Silver & Holman, *Indirect exposure to the September 11 terrorist attacks: Does symptom structure resemble PTSD?*, 21(1) J Traumatic Stress 30 (2008).
[151] *Id.* at 37.
[152] Dirkzwager, Bramsen, Adèr & van der Ploeg, *Secondary traumatization in partners and parents of Dutch peacekeeping soldiers*, 19(2) J Fam Psych 217 (2005) at *7.

(c)

PTSD secondary to witnessing the death or injury to a relative is compensable under the no-fault act.

A no-fault insurer is required to provide payment for injuries "arising out of the ownership, operation, maintenance or use of a motor vehicle." MCL 500.3105(1).

PTSD from witnessing the death or injury of a relative in an auto accident "arises out of" the "operation . . . or use of a motor vehicle . . ." It should, therefore, be compensable under personal protection insurance.

RELIEF REQUESTED

Amicus curiae Michigan Association for Justice, through its attorney Barbara H. Goldman, respectfully asks that this honorable court AFFIRM the March 8, 2011 opinion of the Court of Appeals.

Barbara H. Goldman w/permission by

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[153]Litz, Orsillo, Friedman, Ehlich, & Batres, *Posttraumatic stress disorder associated with peacekeeping duty in Somalia for U.S. military personnel*, 154(2) American J Psychiatry 178 (1997).
[154]*Id.* at *5.